diagnosis of malignant mesothelioma is not clearly established. For this purpose, a study of needle biopsy pleural specimens of suspected cases of malignant mesothelioma compared to larger tissue samples obtained by surgery from the same patients needs to be done.

We appreciate Dr. Kwee and Dr. Utama's interest in our report and thank them for their thought-provoking comments.

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Removal of Foreign Bodies in Children

To the Editor:

We read with interest the article "Foreign bodies in the Tracheo-bronchial Tree" by Weisberg and Schwartz (Chest 1987; 91:730-33). These authors used a rigid bronchoscope for removal of foreign bodies and in their experience, the flexible fiberoptic bronroscope (FOB) proved inadequate in the few occasions when they tried it. Recently, we successfully removed two foreign bodies (plastic whistles) from two 13-year-old boys by using a flexible fiberoptic bronchoscope (Olympus BF, 2TR) with the help of an alligator biopsy forcep.

We agree that removal of foreign bodies may be more easier and possibly also safer with rigid bronchoscopes. However, as flexible fiberoptic bronchoscopes are being used increasingly by medical physicians, an occasional attempt in suitable cases may be worthwhile. We certainly do not recommend this procedure as the method of choice in small children where the FOB cannot be passed easily through the oral or nasal route. Other workers have also used FOB for removal of foreign bodies.1-3

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Metal Fume Fever and Asthma

To the Editor:

We read with great interest the paper, "Occupational asthma due to fumes of galvanized metal" by Drs. Malo and Cartier (Chest 1987; 92:375-77). We have encountered a similar case of bronchial asthma caused by inhalation of metal (zinc) fume.

In October, 1986, a 40-year-old man was hospitalized because of dyspnea and wheezing. He had been a plumber/welder for four years; four months prior to admission, he had a fever (39 to 40°C) and chills five to six hours after exposure to fumes of galvanized metal and he also developed cough, chest tightness, and wheeze in the early next morning. Not only the patient but also his co-workers had experienced symptoms of fevers, chills, malaise, and nausea several times a year, but that was the first time he noted signs and symptoms of bronchospasm. Although he left work due to coughing and dyspnea, he complained of dyspnea and wheeze on exercise.

Skin test showed immediate reactivity to house dust while RAST score was negative. Pulmonary function tests revealed an obstructive ventilatory disturbance, the FEV1 of 2.31 L (74 percent predicted) and FEV1/FVC of 67 percent. FEV1 was increased to 2.97 L after administration of beta-2 adrenergic bronchodilator. Mild bronchial hyperresponsiveness to methacholine was present as Dm20 was 3.1 units. Pulmonary diffusing capacity was 106 percent predicted. Provocation test with galvanized fumes was not performed in our patient, but it is suggested that metal (zinc) fume is an etiologic agent of asthma.

As Drs. Malo and Cartier pointed out, we could not have found any case reports of asthma induced by inhalation of metal (zinc) fume either. However, Amdur et al4 described that chest pain, difficult breathing, and reduced vital capacity are among the symptoms of metal fume fever. In a review article, Mueller and Seger5 mentioned that dyspnea, wheezing, and diffuse cracks may be present in patients with metal fume fever. They also described that an immune complex reaction to the inhaled metal oxide fumes appears to be the most widely accepted theory on the pathogenesis of metal fume fever. Some suggest that the inhaled particles result in inflammation of respiratory tract tissue and release of histamine or histamine-like substances. Another theory proposes a direct toxic effect of the bronchi and pulmonary parenchymal tissues as an etiology of metal fume fever.5

Polymorphonuclear leukocytosis is often associated with metal fume fever, and fever is thought to be mediated by leukocytic pyrogen. The release from activated neutrophils of prostaglandins and leukotrienes as well as airway epithelial damage and release of histamine or histamine-like substances might be responsible for the development of asthma in persons exposed to metal (zinc) fume. Metal fume fever is one of the few ancient occupational diseases still commonly encountered in modern industrial practice.6 Bronchial asthma due to metal (zinc) fume should be added to the literature on occupational asthma.

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To the Editor:

The case report by Kawane and coworkers is interesting. The history, with symptoms and evidence of airway obstruction in
addition to fever, is similar to our case no. 1, who showed a combination of occupational asthma and metal fume fever or hypersensitivity pneumonitis. However, it should be made clear that we may face two distinct conditions with, on occasion, an overlap: 1) metal fume fever, which has never been well characterized from the functional and etiologic points of view; and 2) occupational asthma due to fumes of galvanized metal and possibly related to the presence of zinc in the process. Indeed, our case no. 2 did not show fever or leucocytosis. However, a late bronchospastic reaction was identified.

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Ventilatory Support

To the Editor:

The recent editorial by Duncan et al (Chest 1987; 92:390-92) proposes that inverse ratio ventilation (IRV) may really be PEEP in disguise, and as such may result in associated complications without being fully recognized.

There is little doubt that IRV is an alternate method of obtaining an end expiratory lung volume (therefore PEEP), and I have been addressing this fact over the past two years. The reason this PEEP effect may remain disguised to some results from a lack of familiarity with pressure-controlled ventilation techniques and monitoring. Typical volume-controlled IRV can only be achieved by slowing the flow rate or adding an end-inspiratory pause, and ventilators commonly employed to do this have no method of monitoring end-expired pressures. Current investigation of IRV uses pressure-controlled, time-cycled modes (pcIRV) which allow much greater precision and monitoring when prolonging inspiration (Fig 1). Comparison of these two methods using animal models has already been done which clearly document the superiority of pressure-controlled ventilation. Dr. Duncan's reference to Cole and Sykes comparison of IRV to PEEP must be interpreted with caution as their method employed volume ventilation. It seems that all further reports in the literature must be clear on this point so that we do not end up comparing apples and oranges. In addition to the routine monitoring of hemodynamics, tidal and minute ventilation and mean and peak airway pressures, flow and pressure curves are graphically monitored breath by breath during pcIRV, and an inspiratory pause hold is routinely applied to measure system PEEP. Pressures of 6 to 18 cm H2O are commonly noted by this method which automatically occludes the expiratory circuit at end exhalation using the very method, developed by Pepe and Marini, to which Dr. Duncan refers in his editorial.

Figure 1. Methods of increasing the I:E ratio.

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